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AEROMEDICAL ISSUES RELATED TO POSITIVE PRESSURE BREATHING FOR +G_Z PROTECTION

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This paper has been reviewed and is approved for publication.

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Positive pressure breathing as a method to enhance aircrew tolerance for repeated and sustained high +G_z exposures (PBG) was first considered many years ago. Use of Combined Advanced Technology Enhanced Design G Ensemble (COMBAT EDGE) as a G-protection measure increases overall tolerance to +G_z acceleration by reducing the fatigue associated with performing straining maneuvers during extended or repeated exposures to acceleration stress. Physiologically, PBG exerts its effects upon both the cardiovascular and respiratory systems. The principal benefit of PBG is the maintenance of elevated intrathoracic pressure with minimal voluntary effort on behalf of the individual. Some confusion exists with regard to the potential impact COMBAT EDGE may have on the physical health of aircrew. A certain unknown probability exists suggesting acute and/or long-term exposure to PBG may produce certain undesirable changes in the cardiopulmonary physiology of a pilot. This issue has resulted in the need to provide information regarding the current state of our understanding. Described herein are the principal physiologic issues raised concerning use of COMBAT EDGE, looking to the available literature, past experience, and current and future research answers.

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AEROMEDICAL ISSUES RELATED TO POSITIVE PRESSURE BREATHING FOR $+G_z$ PROTECTION

Development of the Combined Advanced Technology Enhanced Design G Ensemble (COMBAT EDGE) as a G-protection measure began in earnest in 1988. A confounding variable in our attempt to address the physiological impact of COMBAT EDGE is our inability to completely partition out the effects induced by acceleration alone and the interactive coupling effects of acceleration and positive pressure breathing (PPB).

Acute and/or chronic changes (adaptations) to an environmental stress are expected to occur in normal human physiological systems. Acceleration is a severe environmental stress, perhaps the most physically demanding of all the environmental stresses; albeit, likely to have the briefest continuous dose exposure levels. Countermeasures to G-stress provide pilots the ability to more effectively complete their mission by reducing the proportional amount of attention directed at tolerating the G-stress alone. The anti-G straining maneuver (AGSM), existing in one form or another for at least 50 years, is itself a severe physiological stress. However, the risks of performing this maneuver during G-stress are accepted considering the outcome if the pilot performed no procedures to overcome the effect of the stress.

Positive pressure breathing for $+G_z$ protection (PBG), or COMBAT EDGE, is a relatively new arrival in countermeasure technology. Because of its contemporary nature there may be physiologic effects imposed by the technology that are not yet known. However, pressure breathing has been used in aircraft emergency hypobaric situations for at least 40 years. Experimental studies have been, and will be, conducted to reduce the probability of any untoward effect. It is important to remember, PBG was developed to reduce the work (physiological stress) of the pilot during increased G-stress conditions. The tradeoff between decreasing the level of the AGSM and availability of PBG will likely produce a net reduction in the acute and/or chronic adaptive physiological responses. Furthermore, it is important to keep in perspective that peak and sustained high levels of $+G_z$--those requiring high levels of PBG--are infrequent. The total dose exposure might amount to a few hours over the career of the high performance aircraft pilot.

While many of the expressed concerns are legitimate, they have been, are currently, or should be, addressed through animal and human experimental research. There are certain occupational concerns not quantifiable by experimental research designs. These issues are best observed by health surveillance procedures. Furthermore, experimental research related to countermeasure technology has principally involved normal physiology. That is, medical conditions (clinically silent or waivered) present in operational pilots, and hypothesized as potential safety issues, are not always clearly answerable. This underscores the need to investigate this area, beginning with the basic issues related to waivered conditions and their interaction with acceleration stress.

Following is a matrix identifying the principal physiologic issues raised concerning use (acute and chronic) of COMBAT EDGE. Individual issues are placed into three categories: cardiovascular, respiratory, and ophthalmic/otic. Accompanying this chart are annotations describing, in brief, the current state of our understanding regarding the issue(s) looking to the available literature and experience, current research protocols. or future experimental research for the answers.

AEROMEDICAL ISSUES RELATED TO POSITIVE PRESSURE BREATHING FOR $+G_z$ PROTECTION

a) Right Ventricular Distention/Hypertrophy:

Available Literature: A recent Advisory Group for Aerospace Research and Development (AGARD) report, suggested evidence of right ventricular dilatation in French Mirage 2000 pilots. This report initiated a concern within the aeromedical community regarding the influence PBG might have on heart structure. It is important to highlight the point here that the French report did not consider what, if any, influence PPB may have had in these pilots. Conclusions were drawn from the perceived influence $+G_z$ exposure alone caused in this sample of pilots. Noteworthy, is the reported echocardiographic data for right heart dimensions in this study indicating there was no difference between control (age-matched tanker/bomber pilots) and Mirage pilots. The variable receiving the focus of attention in this report was a physiologically meaningless Q-index -- calculated from multiple echographically measured dimensions. Thus, to suggest the Mirage 2000 data provided any insight into the influence PPB has on heart physiology is inappropriate. Notwithstanding, G-only conditions produce dynamic changes in the right side heart volumes. Increased G-stress decreases the volume in both ventricles of the heart due to the hydrostatic pressure effects on blood, sequestering it in the dependent regions of the body. When G-stress is removed, blood, previously sequestered in the dependent region of the body below the heart, returns to the central circulation over the next several heart beats. This process may result in transient increases in right heart dimensions with the returning fluids in some individuals. The volume load is rapidly accommodated and the right ventricle rapidly returns to baseline dimensions. To cause an adaptive change (e.g., hypertrophy) there must be a cnronic inciting mechanism. There is insufficient data yet available to determine whether or not chronic exposure (career) to high-G alone, not to mention high-G plus PPB, will cause negative changes in "normal" right heart structure.

Current Studies: Data from swine indicate peak esophageal pressure (index for intrathoracic pressure) are slightly lower when using PBG than with strain only. This comes from the need to push harder during the strain to support blood pressure when unsupported by PBG. Central venous pressure follows a similar pattern. Also, peak pulse pressures are nearly identical between PBG and strain conditions. These data argue the absence of a pressure mechanism for right heart dilatation using PBG. Another study, in humans, used upright lower body negative pressure (LBNP) to evaluate acute right ventricular (RV) response during the immediate period following presyncopal exposure to LBNP. Apical 4-chamber echocardiographic images recorded RV areas at baseline, peak stress and throughout recovery. Mean RV area at peak stress decreased by >50% from baseline. The first heart beat following pressure release resulted in a 30% increase in RV area from peak stress value. By the second beat, RV area returned to approximately 90% of baseline, and to pre-LBNP value by the third beat of recovery. Return of the sequestered blood, with release of LBNP, did not result in distention of the right ventricle. Therefore, exposure to $+G_z$ stress is unlikely to incite chronic adaptations.

Future Study: A five-nation North Atlantic Treaty Organization (NATO) AGARD working group was established to investigate the influence of chronic $+G_z$ exposure on heart structure using echocardiography. Data gathered in matched-pair samples (control: bomber/tanker/transport (BBT) vs. high performance jet) of pilots will also be limited to providing information regarding the influence of $+G_z$ only on heart structure. The issue of PPB plus $+G_z$ will not be evaluated in this study. Guidance for this issue will await future experimental study.

b) **Left ventricular hypertrophy:**

Available Literature: Cellular changes (e.g., hypertrophy) in the myocardium result from continual (chronic) pressure and/or volume overloads (increased afterloads). The potential for triggering pathophysiologic changes in left ventricular heart muscle using PBG is no greater than that which exists from exposure to straining G-stress. Similar transient (repeated, short-term) increases in afterload are seen in recreational weight lifting athletes and woodwind and brass instrument players without evidence of left ventricular hypertrophy. However, evidence of "physiologic" left ventricular hypertrophy is present in competitive weight lifting athletes.

Current Studies: Swine data indicate transmural pressures between straining and PBG conditions were not meaningfully different. This implies stress in the heart muscle is probably insufficient to cause hypertrophic changes. The probability of a pilot, waivered for borderline left ventricular hypertrophy, worsening his condition by using PBG is not known. Speculation from existing normal physiologic data indicates the probability of this occurring is, most likely, no greater than that which exists for straining pilots with this condition.

Future Study: The NATO AGARD working group protocol will provide information regarding chronic exposure to $+G_z$ and left heart structure. No additional study is planned at this time to investigate the combined effect of PPB and $+G_z$. Necropsy data from the long-term swine study may provide additional information on this issue.

c) **Increased transmural pressure:**

Available Literature: The literature does not address the specific issue of transmural pressure changes with use of PBG. However, data from the canine model, exposed to a range of stresses to 7 $\pm G_x$ (chest to back), suggests differences in diastolic transmural vascular pressures under high $+G_z$ stress are likely minimized by simultaneous proportionate changes in pericardial pressures. In essence, the heart and great vessels are surrounded by hydrostatic pressure that functions as a perfect G-suit. Further, the hypothesis that the lungs are similarly supported by homeostatic hydrostatic gradients, maintaining essentially constant transmural vascular pressures in the dependent regions of the lungs, is supported by data in both dog and human.

Current Studies: Data from the acute swine study indicate pressure differences across the wall of the myocardium, between straining and PBG conditions, are not different. Central venous pressure and right and left ventricular pressures rise during straining and fall with inspiration (with and without PBG). The observed pressure changes are not produced from within the heart, but from external forces developed by the action of the thoracic and abdominal musculature generated in

support of the heart and great vessels. An increase in intrathoracic pressure, developed from the strain and supported by the PBG counterpressure garment, allows thoracic blood pressure to increase. Peak intrathoracic pressure (estimated by esophageal pressure), central venous and esophageal pressure wave forms are not meaningfully different between PBG and strain conditions. PBG does not increase the peak intrathoracic pressure, but moderates or reduces the magnitude of drop in the blood pressure during the inspiratory phase (hypotensive phase) of the strain.

Future Study: A proposed study using a nonhuman primate model will provide additional information in this area.

d) **Decreased venous return:**

Available Literature: Initially, positive acceleration causes a fall in systemic arterial pressure as a direct result of caudal hydrostatic displacement of blood. With continued G exposure, compensatory physiologic mechanisms (mechanoreceptor and chemoreceptor) plus G-suit inflation increases venous return and cardiac output throughout the next phase of G-exposure. However, preexposure values are not restored during G-exposure, since a certain fraction of the blood volume is sequestered in the capacitance vessels and interstitial spaces of the dependent regions. Echocardiographic data indicate there is a rapid venous return to the right heart in the period immediately following $+G_z$ exposure on the centrifuge. The volume returned following G-exposure is dependent upon several factors; most attributed to between-subject physiologic variability.

Current Studies: Swine data indicate central venous pressures and right ventricular pressures return very quickly toward baseline following the offset of acceleration stress. These early recovery pressures remain slightly above prestress values for several seconds; presumably, to accommodate the returning volume from the dependent regions. Data for right ventricular volume changes immediately following (2-3 heart cycles) G-offset are not available. It is unlikely transient accommodation of the returning blood volume could be an inciting mechanism to cause an adaptive change (hypertrophy). Swine data did not show any evidence of an abnormal venous return following either the gradual-onset run (GOR) or simulated aerial combat maneuver (SACM) profiles. This corroborates data from rapid-onset run (ROR) profiles at sustained 3, 5 and 7 $+G_z$ for 60 s.

Future Study: Current echocardiographic study of acute cardiac responses to sustained acceleration stress on the centrifuge will be extended to include PBG in the future. However, important technological hurdles must be overcome before this study is attempted. One laboratory (1 g) study has been completed and a follow-on to that study is attempting to answer technical concerns before this method is transferred to the centrifuge.

e) **Right-to-left shunt:**

Available Literature: The literature indicates 20-30% of adults possess a probe-patent foramen ovale (PPFO). Hypothesized increases in right atrial pressures during the offset of G-stress may result in right-to-left shunting of reduced oxygenated blood or passage of emboli into systemic circulation. Many thousands

of healthy pilots and volunteer subjects (presumably, 20-30% of these individuals having PPFO) have performed AGSMs without evidence of systemic events resulting from right-to-left shunts. Furthermore, aircrew have been exposed to 80-110 mmHg pressure breathing with thoracic counterpressure, at both ground and altitude, without incident.

Current Studies: Swine data, for 9 $+G_z$ and SACM profiles, indicate transmural pressures for the myocardium are not greater using PBG compared to straining alone. Central venous and right ventricular pressures decrease in proportion to the drop in $+G_z$ and G-suit pressure. Furthermore, mean right atrial pressure did not exceed mean left atrial pressure at any time during or immediately following G-exposure. Additional analysis of the data for beat-to-beat changes may identify individual beat pressure mismatches.

Future Study: A nonhuman primate model has been proposed to further address this issue. Chronically instrumented primates with Doppler probes on right and left upper pulmonary veins, as well as the ascending aorta will assess microbubble formation in systemic circulation.

f) **Mitral valve prolapse (MVP):**

Available Literature: No data are available regarding this issue. Pilots with MVP can be granted a waiver to return to flight status if they perform satisfactorily during medical evaluation on the centrifuge.

Current Studies: No current experimental research is addressing this issue.

Future Study: Swine data indicate myocardial transmural pressures using PBG are no greater than those performing the strain alone. Therefore, it is not likely PBG would produce a greater incidence of events related to MVP than with a straining G-exposure alone. Research designed to specifically address this issue has been proposed for the nonhuman primate model.

g) **Left bundle branch block (LBBB):**

Available Literature: While presence of LBBB is disqualifying for entry into pilot training, aviators with acquired LBBB may be granted waiver following successful evaluation. No information is available from the literature specifically addressing the influence of LBBB on sustained acceleration performance with, or without, use of PBG.

Current Studies: No current experimental research is addressing this issue.

Future Study: A study using a nonhuman primate model has been proposed to address this issue.

h) **Sustained ventricular tachycardia:**

Available Literature: Incidence of cardiac dysrhythmias, including ventricular tachycardia, have been reported during high-G centrifuge training. These dysrhythmias generally are asymptomatic and readily convert to sinus rhythm when the subject returns to 1 g; thus, they are considered benign. The high incidence of

dysrhythmias during centrifugation suggests dysrhythmias also occur in flight; most likely, less frequently than seen during centrifuge training. No data are reported in the literature that document incidence of cardiac dysrhythmias using PBG. However, no noteworthy dysrhythmic activity was observed (pilot electrocardiogram (ECG) recorded) during PBG flight trials in the United Kingdom. Also, no reported incidents occurred during United States trials (pilot ECG not recorded). Swine and human centrifuge exposures with PBG demonstrate similar findings.

Current Studies: No current experimental research is addressing this issue.

Future Study: Additional study is necessary to determine whether or not waiverable ventricular premature beats (VPB) might interact with PBG causing an increased incidence of coupling or sustained ventricular extrasystolies.

i) **Spontaneous pneumothorax:**

Available Literature: Acute, *unprotected* overpressurization of the lungs may result in barotrauma. In animal models (from 1/1,000 to 10 times the weight of the average human) *unprotected lungs* failed between 60-100 mmHg (range 40-100 mmHg). Primary failure was due to alveoli "stretching away" from the inelastic pulmonary vascular sheath. With externally applied equalizing pressure to the thorax and the abdomen, intermittent pressures of the order of 170 mmHg for 5 min resulted in no macroscopic evidence of lung damage. Counterpressure applied around the thorax, as with COMBAT EDGE, protects against overdistention of the lungs. Therefore, unless overdistension of the chest, and hence of the lung tissue, is permitted, there is no reason to anticipate damage when using pressures of the order of 50-100 mmHg*. Currently, high performance aircraft pilots waivered back to duty following recovery from spontaneous pneumothorax perform standard anti-G straining maneuvers generating very high levels (>100 mmHg) of intrathoracic pressure supported only by the musculature. Similar magnitudes of intrathoracic pressure ($\geq$ 100 mmHg) are frequently found during great physical exertion, playing wind instruments, weight lifting, parturition, and so forth. PBG, on the other hand, generates similar peak intrathoracic pressures supported by a "balanced" pressure vest. Therefore, it is reasonable to speculate PBG will not increase the incidence of spontaneous pneumothorax resulting from overpressurization or overdistention. Spontaneous pneumothorax occurring during increased acceleration with COMBAT EDGE should not be considered any more incapacitating than pressure breathing at high altitude. If the hypothesis is that pressure breathing exacerbates the severity of spontaneous pneumothorax, altitude may be more incapacitating. This, because $+G_z$ stress can be quickly unloaded, thereby unloading the pressure. Positive pressure at altitude is maintained until a lower level (altitude) can be attained; thus exposure to the positive pressure exposure is extended.

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* Mechanisms for generating increased blood pressure differ slightly for the AGSM and PBG. The end product, peak generated pressure, is the same regardless of which technique is employed. The AGSM generates pressure from within the thorax. Thus, the chest wall and its contents are supported by thoracic and abdominal muscles. PBG, on the other hand, provides pressure from an outside source. The lungs and chest wall are supported by a counterpressure vest in addition to the abdominal bladder of the G-suit. Inspired air volume and flow are individually regulated by simple closure of the glottis.

Current Studies: Swine data indicate increases in intrathoracic vascular pressures are supported by concomitant increases in esophageal pressure (plural pressure), thus minimizing the potential for large transmural pressure values.

Future Study: A study using a nonhuman primate model has been proposed to address this issue. Chronically instrumented animals will provide measures of intrathoracic pressures along the gradient of the lung from apex to base.

j) **Air embolism:**

Available Literature: Most of the literature addresses air embolism as a consequence of diving accidents (usually due to breath holding during ascent) wherein air is released into the pulmonary vasculature. Prior studies, with cadaver and canine models, without use of any chest counterpressure at 1 g, reported evidence of subcutaneous emphysema at pressures of 8.0 and 7.8-8.0 cm H_2O, respectively. No reports of air embolism have been reported to date during experimental use of PBG, nor is air embolism expected to occur with counterpressure at current exposure pressures.

Current Studies: No current study is designed to determine presence of air embolism or subcutaneous emphysema during use of PBG.

Future Study: None planned.

k) **Pulmonary hypertension:**

Available Literature: Pulmonary artery pressure measurements during exposure to increased levels of acceleration using PBG are not available. However, pulmonary artery pressure data are available when conditions are not confounded.

Acceleration: Mean pulmonary arterial and venous pressures in the middle and lower regions of the lung are minimally influenced by increased G-stress. The percentage of the upper lung not perfused increases with increasing G's. Therefore, there is a progressive rise (higher with increasing $+G_z$) in pulmonary arterial pressure (hydrostatic effect) with distance below the nonperfused region of the lung. The abdominal bladder of the G-suit elevates the diaphragm and mediastinum, thus mechanically elevating mean pulmonary pressure by a proportionate amount.

Pressure breathing: Available data indicate pulmonary artery pressure increased in response to the applied positive pressure. However, increases in mean pulmonary artery pressure are not directly proportional to (slightly less than) increases in the applied breathing pressures. Application of chest counterpressure did not meaningfully alter these responses. Pulmonary artery transmural pressure (arterial pressure standardized to esophageal pressure) decreased at all positive breathing pressures. Furthermore, these pressure decreases were more pronounced at higher pressures.

Current Studies: Swine model data under high G conditions indicate no meaningful right ventricular pressure differences between suit-only and PBG conditions. The magnitude and direction of the changes for these variables were similar under the two conditions. Furthermore, peak left ventricular pressure was slightly lower when animals were given PBG compared to non-PBG profiles at 9 $+G_z$. Esophageal

pressure was slightly lower during PBG treatments, as well. These data suggest, at least in the swine model, pulmonary hypertension will not be exacerbated by the use of PBG.

Future Study: A nonhuman primate model has been proposed to further address this issue. Chronically instrumented primates will be used to evaluate pulmonary vascular resistance, right ventricular power, and wall stress using PBG and strain-only conditions.

l) **Microvascular changes:**

Available Literature: No data were available from the literature specifically addressing the issue of microvascular changes with use of PBG.

Current Studies: No current research is addressing this issue.

Future Study: No experimental research proposals addressing this issue are contemplated at this time.

m) **Retinal damage:**

Available Literature: No data were available from the literature.

Current Studies: No ongoing research proposal is investigating the potential for retinal damage during use of PBG in a high sustained acceleration environment

Future Study: The remote possibility of retinal vein thrombosis or other trauma to the retina has been hypothesized. An investigation to measure intraocular pressure and to visualize the retina before, during and after exposure to positive pressure breathing in the laboratory has been proposed. Data have not been collected. It is important to remember that vascular pressure at eye level is generally less than normal during sustained $+G_z$. Normal (1 g) vascular pressure can occur only with performance of an efficient AGSM with or without PBG.

n) **Vestibular dysfunction:**

Available Literature: No data are available to indicate whether or not PBG will have any role in causing vestibular dysfunction. No occurrence of vestibular dysfunction has been reported from several hundred pilots and volunteer subjects exposed to PBG. Only a single incident of vestibular dysfunction related to G is known, and this occurred during a high $+G_z$ only experiment. The subject was exposed to several high-G profiles in a reclined seat (60° seat-back-angle). After several G exposures in this position, the subject complained of severe coriolis. He rested for some time in the gondola and testing resumed only to be terminated with continued symptoms. Recovery was unremarkable in approximately 2 weeks.

Current Studies: No ongoing research protocol is specifically investigating this issue.

Future Study: No experimental research proposals addressing this issue are contemplated at this time.

RESEARCH PROTOCOLS

(December,1993)

Current:

$+G_z$ Alone:

Celio P, et al. *Echocardiography in Humans on the Centrifuge*

Krock LP, et al. *Influence of Specific Resistance Training Exercise on Enhancing Duration Tolerance to Acceleration Stress*

Morgan TR, et al. *Effects of G-layoff on Subsequent Tolerance to $+G_z$.*

Self DA, et al. *Effects of Interruption to an Operational G Exposure Regimen.*

Vandenbosch P, et al. *Echocardiography in NATO Aircrew* (NATO AGARD WG 18).

APPB Related:

Burns JW, et al. *Acceleration Physiology Using the Miniature Swine as a Human Analog.*

Marshall JA, et al. *Laboratory Investigation of the Cardiac Effects of Positive Pressure Breathing Using Echocardiography.*

Travis TW. *Incidence of Acute Adverse Health Effects in Pilots Using a Positive Pressure Breathing Anti-G System.*

Proposed:

Latham, RD, et al. *Cardiopulmonary Dynamics During +Gz Stress with Positive Pressure Breathing and a Simulated Anti-G Strain Maneuver in Primates.*

Krock LP, et al. *Physiological Effects of Assisted Positive Pressure Breathing in Humans During High-G Exposure.*

Samn S, et al. *Mathematical Modeling of Positive Pressure Breathing During G.*

Self DA, et al. *An Investigation Using an Open-loop Animal Preparation that Simulates the Hemodynamic Situations Seen Under Acceleration to Answer Questions that Cannot be Answered in the Intact Animal.*